Suicidal Chlorate Poisoning Treated with Peritoneal Dialysis

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Recovery from chlorate poisoning is rare, and only one case in which dialysis was used has been recorded (Klendshoj *et al.*, 1962). We report here the successful management of a woman who took 40 g. of sodium chlorate in a suicide attempt.

Case Report

The patient, a married woman aged 35, had no history suggestive of renal disease but had a long history of depression. In 1954 and in October 1964 she had been treated for drug overdoses, and in October 1965 for attempted suicide by cutting her wrists. She remained under treatment for three months and was then thought to be well enough to go home. She continued to attend as an outpatient for electric convulsion, which was given at 9 a.m. on 20 December 1965. She walked home alone and took two tablespoonfuls (about 40 g.) of sodium chlorate bought as a weed-killer. She was admitted to Joyce Green Hospital under Dr. P. C. Farrant at 11.15 a.m. Her condition was good, but she began to vomit after arrival. She looked cyanosed and her plasma showed methaemoglobin. She was given methylene blue and vitamin C, and 3 pints (1,700 ml.) of blood were infused and 2 pints (1,140 ml.) removed. She vomited persistently, but by the evening her plasma potassium had risen to 5.4 mEq/l. and her blood urea to 60 mg./100 ml. During the first 18 hours she passed only 90 ml. of urine, and was therefore transferred to Guy's Hospital the next day. Her haemoglobin was 13.5 g./100 ml. and packed cell volume 46%.

On arrival during the afternoon of 21 December her condition had deteriorated. She was a khaki colour, with slate-coloured lips and peripheral cyanosis. There were old scars and recent scratches on her wrists and neck. Her pulse was 88/min., blood pressure 120/90 mm. Hg, and respiration rate 22/min. There were no other physical signs. Blood was immediately taken, and had an extraordinary appearance; the plasma was dark opaque muddy brown and the red cells appeared black and shiny like coal dust. Her blood haemoglobin was 10.7 g./100 ml., and spectroscopy showed free haemoglobin, oxyhaemoglobin, methaemoglobin, and methaemalbumin in the plasma. The oxygen saturation was 82% and standard bicarbonate 26 mEq/l. A blood count showed 4% reticulocytes, a white cell count of 52,800/cu. mm., and a platelet count of only 40,000/cu. mm. Though she was anuric, her blood urea was still 60 mg./100 ml.

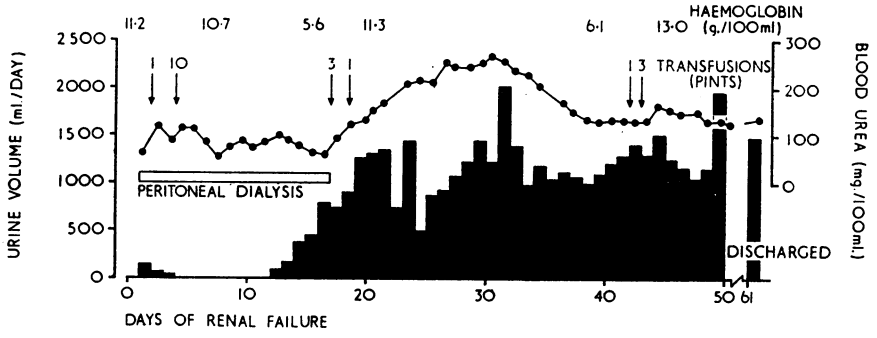
It was thought that any blood transfused would immediately been oxidized and haemolysed, as her own blood had been, so it was decided to perform a peritoneal dialysis to remove the chlorate before transfusing. An intensive dialysis was begun, being continued (Cameron, 1966) for 24 hours before a 10-pint (5.7-litre) exchange transfusion was started. Throughout these procedures she remained in generally good condition but completely anuric. Her mood seemed normal and she no longer complained of depression. Her subsequent progress is summarized in the Figure.

Her plasma rapidly became free of pigments and was completely clear after six days. Her course was complicated by a number of infections, summarized in the Table. Throughout the whole period of the illness, however, her white blood count remained about 20,000 to 50,000/cu. mm., with a marked left shift even in the absence of serious infection, and continued to be high until day 40.

After a partial diuresis her blood urea remained above 100 mg./100 ml., and after nine weeks in hospital she was discharged with a creatinine clearance of 40 ml./min., urea clearance 9 ml./min., blood urea 118 mg./100 ml., and serum creatinine 4.6 mg./100 ml. When seen at the outpatient department in April she was well, with a blood urea of 46 mg./100 ml., and admission was arranged in May for a full assessment. At that time (five months after admission) there was nothing to find on examination, the blood pressure being normal. Intravenous pyelography showed nothing abnormal, but the creatinine clearance remained low (58 ml./min., serum creatinine 1.3 mg./100 ml.), though the blood urea was normal (32 mg./100 ml.). The urine was sterile and concentration was slightly impaired (1018). Renal biopsy was thought not to be justified at that time.

Discussion

Almost all data on chlorate poisoning concern potassium chlorate rather than sodium chlorate, because of extensive industrial and pharmaceutical use of the potassium salt until about 20 years ago. Witthaus (1911) was able to document



The clinical course of the patient.

143 cases from the literature then available; Cochrane and Smith (1940) reviewed only a further eight patients, and since publication of their paper 13 cases have been reported (Gordon and Brown, 1947; Oliver *et al.*, 1951; Klendshoj *et al.*, 1962). Ten of these are included in a review of sodium chlorate

Infections

Day	Site	Organism	Antibiotics
1–5	—	—	Benzylpenicillin 500,000 u. q.d.s.
2–4	Lung	?	
2–4	Urine	*Str. faecalis*	
3–14	—	—	Ampicillin 250 mg. q.d.s.
5	*Peritoneum	Atypical *E. coli*	—
7–12	Urine	*Str. faecalis. E. coli.* Proteus	—
12–22	Mouth	*E. coli*	
12	*Peritoneum	Paracolon	
14–19	—	—	Chloramphenicol 1 g. q.d.s.
18–20	*Peritoneum	*E. coli.* Yeast	
18–20	Urine	*E. coli*	
18–22	Lung	*E. coli*	
19	†Blood	*E. coli*	
19–25	—	—	Cephaloridine 0·5 g. q.d.s.
22	Mouth	*E. coli.* Paracolon	—
22–27	—	Candida	Nystatin lozenges
24–27	Urine	*E. coli.* Haemolytic strep.	
26–34	—	—	Ampicillin 250 mg. q.d.s.
32–34	Lung	?	—

* Only on days 18–20 was the dialysate turbid.
† This was the only positive blood culture of many taken.

poisoning by Dérot *et al.* (1948). Apart from these only three case reports (Jacobi, 1879; Stryjzowski, 1931; Klendshoj *et al.*, 1962) refer to sodium chlorate poisoning. The sodium salt may be expected to be even more toxic than the potassium salt, because it contains more chlorate on a weight-for-weight basis (77% as compared with 68%) and is 16 times more soluble in cold water.

The fatal dose of chlorate is usually 20–30 g. (Witthaus, 1911). The smallest dose causing the death of an adult is 7.5 g. (Bernstein, 1930) taken in a tooth-paste by a suicidal Army officer; and the largest dose with survival is 47 g., taken in divided doses over 13 hours (Gesenius, 1882). Previous renal impairment, as in the patient of Cochrane and Smith (1940), would decrease the fatal dose, since 95% of the drug is eliminated in the urine. Few patients survive chlorate poisoning. Blyth (1884) collected 39 cases—28 fatal—and of Witthaus's 143 patients 116 died. The average survival was just over four days, ranging from four hours to 12 days. Renal failure was recorded early. The unfortunate Dr. Fountain took 29.2 g. of potassium chlorate as an experiment to prove its harmlessness in 1858 and died "on the seventh day of nephritis" (Jacobi, 1860).

The symptoms and signs of chlorate poisoning have been well described. After ingestion there is usually a period of one to four hours before vomiting begins, often accompanied by diarrhoea and abdominal pain. Dusky cyanosis and dyspnoea follow, the blood pressure tends to fall, and the heart beat to become irregular. The liver and spleen may be enlarged and tender. The urine is scanty or even absent, is brown or black in colour, and contains casts, red cells, free haemoglobin, and methaemoglobin. The blood is brownish in colour and the plasma contains free haemoglobin and free methaemoglobin. The red count is very low and the white cell count persistently high. Agitation, muscular weakness or twitching, occasionally fits, and a falling blood presure lead to coma and death. Many of these symptoms and signs undoubtedly arise from uraemia and hypoxia. At necropsy the kidneys are brown in colour, hyperaemic, and larger than normal. There are signs of gastro-enteritis, the liver and spleen are enlarged, and the blood is chocolate-coloured.

Apparently chlorate is toxic because it is such a powerful oxidizing agent. It attacks all cells, including liver, kidney, and the circulating red cells (Richardson, 1937). SH groups would be particularly vulnerable. The red cells are largely haemolysed and the released haemoglobin converted to methaemoglobin. The intact red cell has considerable capacity to reduce methaemoglobin to haemoglobin (Jaffé, 1964), but this mechanism presumably cannot operate after haemolysis, and methaemoglobin accumulates. This suggestion is confirmed by Gordon and Brown (1947), who noted that the haemoglobin in the washed red cells of their patient was virtually all true haemoglobin, whereas the free pigment was mostly methaemoglobin. The dangers to the patient are therefore direct tissue toxicity, tissue hypoxia, and haemolysis. The haemolysis could liberate enough free potassium to make the plasma K^+ concentration rise to lethal levels, and this presumably accounts for some of the very rapid deaths. Any patient who goes into immediate renal failure will retain almost all the absorbed dose indefinitely, so that dialysis becomes essential for its removal. The chlorate in the dialysate was not measured, but it is known to be freely dialysable from blood and from tissue in vitro (Witthaus, 1911).

The only other case in which dialysis was performed was that reported in detail by Klendshoj *et al.* (1962) and mentioned by Schreiner (1958) and Doyle (1962). The patient was a 28-year-old Korean physician who took exactly 40 g. of sodium chlorate in mistake for sodium chloride in a laboratory experiment. Haemodialysis was performed on the second day to remove chlorate, and on the eighth and fourteenth days because of renal failure. In addition, an 18-pint (10-l.) exchange transfusion was carried out during the first dialysis. The authors attributed this recovery to prompt exchange transfusion and dialysis. A haemodialysis was begun on one patient (Dérot *et al.*, 1948) but was abandoned after 30 minutes because of clotting problems.

Normally, patients with acute renal failure may be expected to recover renal function completely. The failure of our patient to recover completely presumably reflects some irreversible damage due to direct toxic effect on the renal parenchyma, as well as reversible changes due to the haemoglobinuria. One can only guess whether the peculiar susceptibility of our patient to infection (despite excellent control of blood urea on continuous dialysis) was related to some specific feature of chlorate poisoning.

ADDENDUM.—It has been drawn to our attention that Case 10 of the paper by Pringle and Smith (1964) refers to a 17-year-old youth with renal failure from sodium chlorate poisoning who was treated with peritoneal dialysis for 14 days and who recovered.

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